## Trophoblasts Regulate the Placental Hematopoietic Niche through PDGF-B Signaling.

**Journal:** Dev Cell

**Publication Year:** 2012

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**PubMed link:** 22387002

**Funding Grants:** UCLA CIRM Research Training Program II

**Public Summary:**
The placenta is a hematopoietic organ that supports hematopoietic stem/progenitor cell (HSPC) generation and expansion without promoting differentiation. We identified PDGF-B signaling in trophoblasts as a key component of the unique placental hematopoietic microenvironment that protects HSPCs from premature differentiation. Loss of PDGF-B or its receptor, PDGFRbeta, induced definitive erythropoiesis in placental labyrinth vasculature. This was evidenced by accumulation of CFU-Es and actively proliferating definitive erythroblasts that clustered around central macrophages, highly reminiscent of erythropoiesis in the fetal liver. Ectopic erythropoiesis was not due to a requirement of PDGF-B signaling in hematopoietic cells but rather in placental trophoblasts, which upregulated Epo in the absence of PDGF-B signaling. Furthermore, overexpression of hEPO specifically in the trophoblasts in vivo was sufficient to convert the placenta into an erythropoietic organ. These data provide genetic evidence of a signaling pathway that is required to restrict erythroid differentiation to specific anatomical niches during development

**Scientific Abstract:**
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